

## REOPERATION AFTER THE ARTERIAL SWITCH OPERATION FOR TRANSPOSITION OF THE GREAT ARTERIES

Although most children after an arterial switch operation for transposition of the great arteries have normal development and cardiac function, a few require reoperation. During the last 10 years, 68 of 753 patients who underwent arterial switch operations (9.3%) underwent 75 reoperations. Thirty underwent early reoperation (<30 days or during the same hospital stay) and 38 underwent late reoperation. Causes for reoperation included pacemaker insertion ( $n = 5$ ), left diaphragm plication ( $n = 4$ ), revision for hemostasis ( $n = 1$ ), mediastinitis ( $n = 2$ ), superior vena cava thrombosis ( $n = 9$ ), subvalvular pulmonic stenosis ( $n = 5$ ), supraaortic pulmonic stenosis ( $n = 16$ ), residual atrial ( $n = 2$ ) or ventricular ( $n = 8$ ) septal defects, isolated mitral valve insufficiency ( $n = 2$ ), aortic valve insufficiency (either isolated [ $n = 1$ ] or in association with mitral incompetence [ $n = 1$ ] or stenosis [ $n = 1$ ]), left coronary artery ostial stenosis ( $n = 1$ ), and recurrent aortic ( $n = 6$ ) or neo-aortic ( $n = 4$ ) aortic coarctation. In all but 27 patients, the residual defects were already present immediately after the completion of the arterial switch operation; however, only patients with critical lesions were reoperated on early. Interventional catheterization procedures were performed when indicated; however, they only postponed inevitable reoperation. Successful relief of superior vena cava thrombosis was achieved by atriojugular bypass grafting in two patients, by early open thrombectomy in six patients, and by direct patch angioplasty of the superior vena cava once. Patch plasty for subvalvular or supraaortic pulmonic stenosis was carried out in 21 patients, septal defect closure was carried out in nine patients, and pulmonary artery banding was performed in one patient with criss-cross atrioventricular relationship and multiple ventricular septal defects. Valve repair was performed in all five patients with either isolated or combined aortic and mitral valve dysfunction. One patient with left coronary ostial stenosis underwent a patch enlargement of this ostium. Recoarctation was repaired by end-to-end anastomosis in eight patients and by a subclavian flap and a patch angioplasty in one patient each. Seven patients underwent a second reoperation for supraaortic pulmonary stenosis ( $n = 3$ ), mitral valve replacement ( $n = 1$ ), ventricular septal defect closure ( $n = 1$ ), and recurrent coarctation ( $n = 2$ ). There were six intraoperative (8.8%) and two late deaths. All early deaths occurred after early reoperations. Risk factors for intraoperative death at reoperation were early reoperation ( $p < 0.01$ ) and multiple residual ventricular septal defect ( $p < 0.01$ ). Among the entire group who underwent arterial switch operation, there were no risk factors for the overall group of persons undergoing reoperation; however, univariate analysis revealed risk factors for reoperation for right ventricular outflow tract obstruction. These included nonneonatal repair ( $p < 0.01$ ), long-standing pulmonary arterial banding ( $p < 0.01$ ), associated defects

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( $p < 0.001$ ), and the surgical technique used for pulmonary arterial reconstruction (single versus two pericardial patches, direct anastomosis without patch insertion;  $p < 0.05$ ). Multivariate analysis revealed that only the presence of a hypoplastic native aortic anulus as opposed to the native pulmonary anulus was a risk factor for postoperative pulmonary stenosis and reoperation. Mean follow-up of  $70 \pm 19$  months was achieved in all survivors, and they were all free of symptoms and need for medication. In conclusion, most lesions requiring a reoperation after an arterial switch operation are detectable early, and intraoperative echocardiography might consequently be useful. Most late reoperations can be prevented by primary neonatal repair of almost all forms of transposition of the great arteries. (J THORAC CARDIOVASC SURG 1995;110:892-9)

The arterial switch operation (ASO) is now recognized worldwide as the procedure of choice for treatment of children born with transposition of the great arteries (TGA) in either simple<sup>1-3</sup> or complex<sup>4,5</sup> forms. Because the ASO is more technically demanding, the initial overall early mortality was higher than for atrial switch procedures; with increasing experience and passing over the learning curve, however, the mortality rates have been lowered and presently vary between 0% and 10% in most series.<sup>6-9</sup> Rates now compare well with those for the atrial switch procedure.<sup>10</sup> In addition to mortality rate, to definitely determine the ASO to be the optimal treatment for TGA, morbidity rates need to be within acceptable ranges. Although most of these children have normal psychomotor development and cardiac function, a few are not free from early or late complications and may require reoperation. Reoperations may be indicated either by residual or recurrent lesions or by the onset of a new acquired lesion related to the procedure. During the last 10 years, since the ASO was introduced at our institution, a total of 68 patients were reoperated on after ASO. This work was aimed at assessing the causes and the results of these reoperations and to determine the existence of any risk factors.

## Patients and methods

Between March 1983 and July 1994, 753 ASOs were performed in our institution for various forms of TGA. The distribution of the different forms according to the age and the results were previously published.<sup>8</sup> Briefly, 68% of patients had an intact ventricular septum and 32% had an associated ventricular septal defect (VSD). Neonatal repair, which is now the rule in the majority of cases, was performed in 83%. Repair beyond the neonatal period was performed in 17% of patients, 70% of whom underwent two-stage correction.

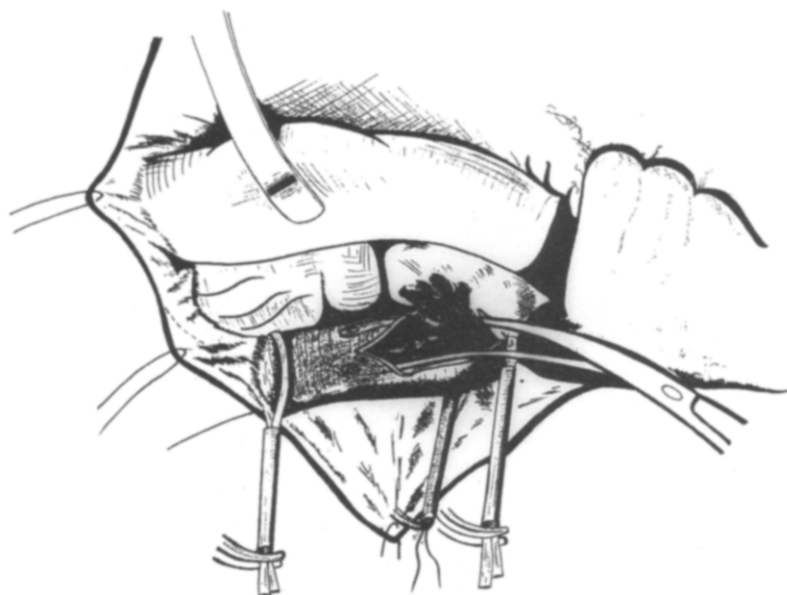
Seventy patients (9.3%) required reoperation. Forty-six

**Table I.** Reoperation after ASO for TGA

Cause of reoperation	Patients	Median delay (mos.)
Pacemaker	5	0.5
Diaphragm plication	4	0.7
Hemostasis	1	0.03
Mediastinitis	2	1.5
SVC thrombosis	9	0.16
RVOTO	21	14
SVPS	16	
SbVPS	5	
Septal defects	10	2
ASD	2	
VSD	8	
Valvular disease	5	24
MI	2	
AI	1	
Combined MI & AI	2	
Coronary artery stenosis	1	117
Left ostium	1	
LVOTO	11	4
SAoS	1	
ReCoA	10	

SVC, Superior vena cava; RVOTO, right ventricular outflow tract obstruction; SVPS, supra-valvular pulmonary stenosis; SbVPS, subvalvular pulmonary stenosis; ASD, atrial septal defect; MI, mitral insufficiency; AI, aortic insufficiency; LVOTO, left ventricular outflow tract obstruction; SAoS, subaortic stenosis; ReCoA, recoarctation.

were male and 24 were female. The median age at reoperation was 4.2 months (range 0.2 to 117 months) and the median delay between ASO and reoperation was 3 months (range 1 day to 116 months). Reoperations were mandated by either residual, recurrent, or acquired lesions on both right and left cardiovascular structures (Table I). Two patients, one with postoperative lateral myocardial infarction who underwent orthotopic heart transplantation in another institution and another who underwent pulmonary artery patch angioplasty in another institution, were excluded from this series. The remaining 68 patients form the basis of this analysis. Also excluded from this study were two patients who were reoperated on for a noncardiac lesion. Patients scheduled for a reintervention in the coming months (two for aortic insufficiency



**Fig. 1.** Superior vena cava thrombectomy. After insertion of a central line through the right atrial appendage, the internal jugular catheter is removed. The superior vena cava is controlled and crossclamped between the innominate vein and its cardiac junction. A longitudinal venotomy is performed, and the thrombus is removed with a right-angle forceps. The venotomy is then closed.

and one for pulmonary stenosis) were also not included in this analysis.

**Definitions.** *Early reoperations* were defined as reoperations performed within 30 days of initial ASO or during the same hospital stay. They were generally carried out because of life-threatening complications and failure of intensive medical support. *Late reoperations* were defined as operations performed after hospital discharge and beyond the first postoperative month; these were generally performed as elective procedures.

**Anatomic findings.** Five patients required pacemaker insertion, four had left diaphragm plication for phrenic nerve palsy, one was reoperated on for revision of hemostasis, and two were reoperated on for mediastinitis.

**Superior vena cava thrombosis.** Superior vena cava thrombosis was the cause for reoperation in nine patients. In all but one, an acute superior vena caval syndrome occurred within two days after operation. In addition to the patent clinical symptomatology, the diagnosis was confirmed by venous Doppler sonographic study and internal jugular opacification. After initially alert to this complication, even after operation for other heart defects, medical therapy with full heparinization was started, in one case associated with urokinase thrombolysis. This approach, however resulted in high morbidity and mortality and in only partial relief of the thrombosis in the survivors. Three patients receiving this therapy after an ASO were reoperated on and underwent atriojugular bypass with a 6 mm polytetraethylene graft\* twice and

direct patch angioplasty of the superior vena cava in one case. This unsatisfactory experience prompted us more recently in a more aggressive approach whenever confronted with this complication. Early open thrombectomy without cardiopulmonary bypass assistance was therefore performed in six patients. The details of the surgical technique are depicted in Fig. 1.

**Right ventricular outflow tract stenosis.** Twenty-one patients were reoperated on for right ventricular outflow tract obstruction. In 16, the stenosis was supra-ventricular, located on the pulmonary suture line. In five others, the stenosis was located at the valvular or subvalvular level. The median preoperative right ventricle-pulmonary artery gradient was 70 mm Hg (range 40 to 140 mm Hg) and the median delay between ASO and reoperation was 14 months (range 1 to 90 months). All patients underwent balloon angioplasty with immediate partial relief of the stenosis; however, stenosis recurred between 2 and 6 months after dilation. None of them underwent stent placement. Surgery was performed with normothermic cardiopulmonary bypass and a beating heart. For supra-ventricular stenosis, a circular polytetrafluoroethylene patch\* was used to enlarge the stenotic area. For valvular or subvalvular stenosis, a transannular patch was inserted after resection of a hypertrophic parietal band. In all cases, the coronary anatomy allowed a transannular incision; an extracardiac conduit would otherwise have been inserted.

**Septal defects.** Ten patients had residual septal defects. In two, the defect was at the atrial level and was closed with a patch with the patient on cardiopulmonary bypass. Indication for reoperation in these patients was dictated

\*Gore-Tex vascular graft, registered trademark of W. L. Gore & Associates, Inc., Newark, Del.

by the presence of a left-to-right shunt higher than 2:1 and distention of the right heart cavities. In eight others, the defect was at the ventricular level. Four of these underwent an attempt at closure of multiple VSDs along with the ASO. Four initially had single VSDs, two of which had been neglected at the primary ASO because it was believed that spontaneous closure would occur and the other resulting from patch deinsertion. Early reoperation was mandatory in all those with multiple VSDs, three of whom had closure with a single large patch as previously described.<sup>11</sup> In the remainder with criss-cross atrioventricular relationship, a pulmonary artery band was placed. All the residual VSDs were documented by color echo Doppler studies and cardiac catheterization. Reoperations were mandated by the inability to wean the patient from ventilatory support or by persistent congestive heart failure with a left-to-right shunt greater than 2:1.

**Left-sided valvular lesions.** Both mitral and aortic valves were of concern. Isolated mitral valve insufficiency occurred twice. In one case it was associated with annular dilatation and a cleft in the anterior leaflet; in the other it was caused by ruptured chords. Two other patients had mitral valve dysfunction associated with other lesions. One had parachute mitral valve stenosis associated at reoperation with a subaortic membrane and aortic insufficiency; the other had an associated severe aortic insufficiency. Techniques for repair were basically adapted from Carpentier's techniques and closure of clefts.<sup>12</sup> None of these patients underwent primary mitral valve replacement. Indications for operation were ongoing congestive heart failure with heart enlargement in two cases and severe mitral incompetence occurring immediately after the ASO, forbidding weaning from ventilatory support. Two of these patients had nonfatal perioperative lateral myocardial infarctions, which probably led to chordal rupture in one and to papillary muscle fibrosis in the other.

A total of three patients had severe and progressive aortic valve insufficiency. In all cases, aortic valve repair was performed after transection of the anterior pulmonary trunk and crossclamping of the aorta. The mechanisms of aortic insufficiency were related in one case to iatrogenic injury of the aortic leaflet during VSD closure through the pulmonary artery and in the two other cases to a dilatation of the noncoronary sinus of Valsalva with a prolapse of the corresponding leaflet. Techniques for repair included commissuroplasty<sup>13</sup> and aortic annuloplasty.<sup>14</sup> In two cases, mitral valve repairs were concomitantly performed.

**Coronary artery disease.** In one patient, cardiac failure developed 3 months after ASO as a result of stenosis of the left coronary ostium. At that time medical therapy was started to control the cardiac failure, and the right coronary system progressively became dominant. There was a positive result of exercise testing for myocardial ischemia on the anterolateral left ventricular wall, however, and the patient was reoperated on at 10 years of age. A patch angioplasty with the anterior wall of the pulmonary artery was performed to enlarge the left coronary ostium.

**Aortic isthmus.** Ten patients had to be reoperated on for aortic coarctation. In four instances, the coarctation process appeared within 3 months after ASO. All of these

patients had documentation of a free unobstructed aortic arch after ASO. In one case, the aortic coarctation, although initially present, was overlooked, and it was only the absence of femoral pulses immediately after operation that prompted early reoperation. In five other patients, recurrent coarctation appeared during the first postoperative year. The coarctation was approached through a left thoracotomy in all but one patient. Extended end-to-end anastomosis was performed in six patients, patch angioplasty was performed in one patient, and two patients underwent subclavian flap angioplasty.

**Second reoperation.** Seven patients underwent a second reoperation. Two underwent a second reoperation for tubular hypoplasia of the pulmonary arteries; both underwent patch angioplasty of the intrapericardial pulmonary arteries from one hilum to the other. A third patient had a persistent mitral insufficiency despite a previous repair and persistent subaortic stenosis. He underwent mitral valve replacement with a CarboMedics 18 (CarboMedics Inc., Austin, Tex.) and a septoplasty procedure.<sup>15</sup> One patient with multiple VSDs and criss-cross atrioventricular relationship finally underwent successful VSD closure 2 years after pulmonary artery band placement. Another still had persistence of multiple VSDs, which were closed 2 years after the reoperation. The last two patients still had recurrent coarctation after patch angioplasty and underwent end-to-end anastomosis.

**Follow-up.** Follow up was achieved for all the survivors by means of telephone calls and contact with referring pediatric cardiologists. After reoperation, the patients were studied twice per year by means of bidimensional echocardiography and Doppler assessment. New cardiac catheterizations were not performed except in case of residual lesions. The mean duration of follow-up after reoperation was  $70 \pm 19$  months, and it was 16.4 after the initial ASO.

**Statistical analysis.** Risk factors for postoperative death and for the likelihood of reoperation at initial presentation were assessed by univariate analysis. Dichotomous variables were analyzed by Fisher's exact test or  $\chi^2$  test and Student's *t* test was used for continuous parameters. Multivariate analysis with SPSS software (SPSS, Inc., Chicago, Ill.) for the Macintosh computer (Apple Computer, Inc., Cupertino, Calif.) was undertaken to define the risk factors for the likelihood of reoperation at initial presentation. Time-related events were analyzed by the Kaplan-Meier methods. Ratios are expressed with 70% confidence intervals.

## Results

**Mortality.** There were six early deaths (8.8%; 4.9% to 13.9%). All six patients underwent early reoperation because of life-threatening residual or iatrogenic defects. Repair failed in two who had "Swiss cheese" multiple VSDs; one had a severe aortic regurgitation resulting from leaflet injury during VSD closure and the other had ruptured chords of the mitral valve. In another patient with extremely poor left ventricular function and residual coarctation, death occurred immediately after un-

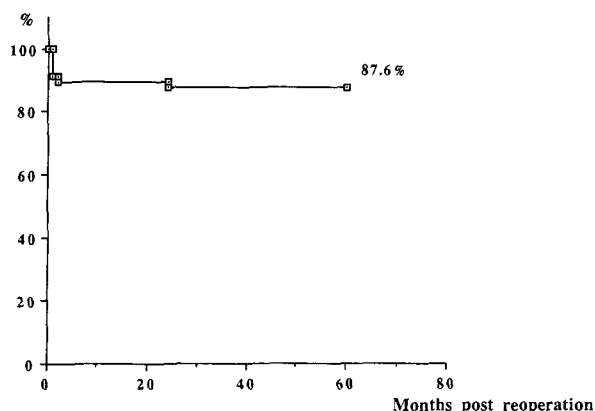


Fig. 2. Actuarial survival curve after reoperation.

clamping of the aorta. The remainder had thrombosed atriojugular bypass for superior vena cava thrombosis.

There were two late deaths in two patients after relief of superior vena cava thrombosis, 6 months and 2 years after reoperation. In one case death was caused by residual multiple VSDs; in the other, it resulted from acute bronchiolitis. All patients who underwent early open thrombectomy of the superior vena cava recovered uneventfully, with relief of symptoms within 48 hours after operation. They were maintained on a regimen of anticoagulation therapy for 3 months. Patency of the superior vena cava was regularly assessed by means of venous Doppler investigation without evidence of recurrence. The single survivor among patients who underwent atriojugular bypass at 3 years after reoperation had a patent graft and has been kept on a regimen of antiplatelet aggregation therapy. No risk factors for superior vena cava thrombosis were found.

There were no deaths in the group of patients reoperated on for right ventricular outflow tract stenosis. Relief of the obstruction was satisfactory in 19 patients, with a mean residual systolic gradient between the right ventricle and the pulmonary artery of 15 mm Hg. Two patients, however, underwent a second reoperation after several attempts at balloon angioplasty. At the last follow-up, all these patients are free of complications without medication.

All the patients who underwent closure of a residual atrial septal defect recovered uneventfully. Four with residual single VSDs are also free of complications. Patients with residual multiple VSDs

had a more difficult postoperative course, with two survivors. One, with associated coarctation and criss-cross atrioventricular relationship, underwent pulmonary artery banding because of the anatomic complexity of the septal structures. He underwent VSD closure 2 years after pulmonary artery banding and had a pacemaker inserted. The other had a stormy postoperative course after secondary closure of residual multiple VSDs, with severe low cardiac output and renal failure. Peritoneal dialysis was instituted and maintained for 10 days, along with maximal inotropic support. This patient eventually recovered and was discharged from the hospital 1.5 month after operation. One year later recovery was complete, with normal echocardiographic indexes under afterload reduction therapy. The single patient reoperated on for coronary ostial stenosis recovered uneventfully and is scheduled for a control coronary angiography within the coming year. Noninvasive investigations showed a satisfactory repair.

Valve repair were assessed by color echo-Doppler studies. All showed a significant reduction of the valvular incompetence concomitant with improved cardiac function. One patient underwent second operation for an association of complex residual mitral valve insufficiency and subaortic stenosis. A mechanical mitral prosthesis was inserted and the left ventricular outflow tract was enlarged by patch septoplasty. Two years after operation, the patient is free of complications on an anticoagulation regimen. All patients reoperated on for aortic coarctation did not demonstrate any residual aortic arch obstruction.

Univariate statistical analysis did not reveal any risk factors for reoperation when considering the entire population of patients undergoing ASO, except for risk factors for occurrence of right ventricular outflow tract obstruction. These were nonneonatal repair ( $p < 0.01$ ), long-standing pulmonary artery banding ( $p < 0.01$ ), associated defects ( $p < 0.001$ ), and the surgical technique for pulmonary artery reconstruction (single versus two pericardial patches, direct anastomosis without patch insertion;  $p < 0.05$ ). Multivariate analysis, however showed that only the presence of a hypoplastic aortic anulus rather than the native pulmonary anulus was a risk factor for the occurrence of pulmonary stenosis. Also risk factors for in-hospital death at reoperation were early reoperation ( $p < 0.01$ ) and multiple residual VSDs ( $p < 0.01$ ). The 5-year actuarial survival after reoperation was  $87.6\% \pm 5.25\%$  (Fig. 2).

## Discussion

Although the ASO is accepted worldwide for anatomic repair of TGA, long-term outcomes remain unknown. In some instances, complications may occur and necessitate either an interventional cardiologic therapy, such as balloon angioplasty or stent placement, or reoperation. In our institution the ASO option was started a decade ago, and follow-up ranges between 1 month and 10 years. Within this time frame, 9.3% of the patients had to be reoperated on for several types of lesions located either on the right side, on the left side, or on the septal structures. Excluding early reoperations, the reoperation rate was 4.7%, with a median delay of 13 months (range 2 to 116 months). Continuous medical attention is therefore mandatory in this population.

Superior vena cava thrombosis has been described in association with several types of pediatric disorder.<sup>16</sup> There are, however, few reports of this occurring after cardiac operations. When reviewing the data from our ASO population, we were not able to find specific risk factors for the occurrence of this complication. We therefore reviewed the data from all neonates undergoing an open or closed heart procedure during the last 5 years, with particular attention to the occurrence of superior vena cava thrombosis. Several hypothetic risk factors were addressed; these included hemodynamic status, sepsis, use and duration of central jugular lines, coagulation status, and continuous cardiopulmonary bypass with or without bicaval cannulation or circulatory arrest. None of these variables came out as a significant risk factor, and particularly the use of internal jugular venous catheter was not found to be associated with an incremental risk for superior vena cava thrombosis. Whatever the cause, which is probably multifactorial, we have found that the only efficient strategy to adopt whenever confronted with this problem was an emergency early open thrombectomy followed by anticoagulation therapy for 3 months. Any delay in surgical therapy may lead to thrombus organization and render thrombectomy ineffective.

Right ventricular outflow tract stenosis has previously been reported as an important complication after ASO.<sup>17,18</sup> Several risk factors have been defined. From our univariate study, four risk factors appear to significantly contribute to the occurrence of this complication, and since our move to a more uniform approach (the single-stage neonatal repair and reconstruction of the pulmonary artery with

a pantaloon-shaped fresh autologous pericardial patch), incidence of this complication has been considerably reduced. Indication for reoperation in this setting remains unclear. Although all but one patient had no or mild symptoms, we empirically decided to intervene when the peak systolic gradient between the right ventricle and the pulmonary artery was 60 mm Hg or above. All these patients underwent balloon dilation several times; however, the recurrence rate was high enough to propose surgical pulmonary angioplasty in 19. No attempt at stent placement was performed during that period. Interestingly enough, all the patients who were reoperated on had relatively early occurrence of pulmonary stenosis within the first postoperative year with progressive accentuation. At present, the single risk factor not neutralized by our current technique is the size mismatch between the aortic and pulmonary anuli, particularly in cases of TGA, VSD, and coarctation.<sup>5</sup> More recently, Akiba and colleagues<sup>19</sup> developed the hypothesis that postoperative modification of the neosubpulmonary flow may be the source of turbulences and right ventricular outflow tract obstruction. Preservation of the pericardial patch with glutaraldehyde is also widely used. In our present series it did not appear to be a risk factor, probably because it was used only in the first 10 patients. We strongly believe, however, that the more viable the pulmonary patch, the lower the risk of late pulmonary stenosis.

Residual VSDs were the most lethal lesions in this series (60% of all deaths). As already reported, the "Swiss cheese" form of multiple VSDs is associated with a higher mortality rate,<sup>11</sup> not only because of the complexity of the repair but also because accurate diagnosis is difficult. Residual VSDs can be the postoperative source of a torrential left-to-right shunt, and reoperation with cardiopulmonary bypass may therefore add to an already deteriorated myocardial function. We have little experience in banding of the pulmonary artery after the ASO in this particular situation. We wonder, however, whether this procedure could not lead to a right ventricular distention and kink the coronary artery at the point of reimplantation. Percutaneous defect closure either before or after operation would represent a significant advance in the management of these patients<sup>20</sup> and might avoid reoperation in several cases.

The fate of the neo-aortic valve has been questioned in previous reports.<sup>21,22</sup> Although at birth aortic and pulmonary leaflets have the same histo-

logic patterns, aortic valve insufficiency occurs with varying rates according to different series and seems to be a more serious problem after conversion of an atrial switch to an ASO.<sup>23</sup> In the population of patients undergoing primary ASO, Ungerleider and coworkers<sup>25</sup> reported a case of aortic valve replacement 10 years after ASO. To the best of our knowledge, there have been no reports of aortic valve repair in this situation. In our series, this was successfully achieved in two patients. The mechanisms of aortic valve insufficiency were related in one patient to severe leaflet injury during transpulmonary VSD closure and in the two others to a dilation of the neononcoronary sinus of Valsalva with adjacent leaflet prolapse. We believe that after operation the region of aortic and coronary sutures are a site of turbulences that may in time cause an enlargement of the aortic root in its weakest portion; that is, the one without suture line scars. We believe that it may be possible to prevent this complication by achieving a single-stage neonatal repair or, when there is an important size mismatch between the neo-aortic root and the distal aorta, by resection or plication of the neononcoronary sinus of Valsalva at the time of the initial ASO.

The mitral valve was also of concern in four cases, twice in association with aortic valve insufficiency. Postoperative myocardial infarction was probably the cause of chordal rupture in one case and anterior papillary dysfunction in another. In the other two patients, the mitral valve disclosed congenital malformations, a parachute mitral valve and a cleft in the anterior leaflet. Although infrequent, mitral valve malformations have been described in association with TGA.

Coronary perfusion has been investigated in different ways after ASO,<sup>26</sup> and in most patients there is excellent coronary blood flow. With the introduction of routine coronary angiography before school age, some abnormalities of the coronary anatomy may emerge. The patient who underwent a patch angioplasty of the left coronary ostium was known to have abnormal left coronary perfusion. Coronary angiography in another patient disclosed a left coronary ostium arising from the pulmonary artery. A very small left coronary artery was not reimplanted at the time of neonatal ASO because it was considered too small. Although this patient has a dominant right coronary system and is free of symptoms, careful follow-up is necessary for possibly another reoperation.

Seven patients have to be reoperated on for aortic

coarctation. In four instances, the lesion was not documented before ASO and appeared within 1 to 3 months after operation. Muster and colleagues<sup>27</sup> relate it to the Lecompte manoeuvre. Our data, however, do not support that hypothesis because in our entire series of patients undergoing ASO who had the Lecompte maneuver, the occurrence rate for neocoarctation was extremely low (below 0.5%). Rather, we believe that in some cases during ductal division exuberant ductal tissues may invaginate within the aortic lumen and cause a secondary aortic coarctation. It has recently been demonstrated that balloon angioplasty for recurrent coarctation results in a significant reduction of the coarcted area and relief of the symptoms and thus should be the first therapeutic approach attempted in this setting.

Although the reoperation rate after ASO is not zero, it is similar to the reoperation rate after atrial switching procedures.<sup>28</sup> Therefore, ASO can be proposed for all forms of TGA on the basis of probability of freedom from reoperation.

In conclusion, as time elapses after the ASO in a small percentage of patients, some long-term complications are emerging. They can occur at every site of the cardiac anatomy, and continuous medical attention is therefore mandatory. Some are avoidable, however, probably by using precise surgical techniques during initial ASO. From the results of this study, some guidelines to avoid late pulmonary stenosis can be proposed: (1) neonatal single-stage repair, (2) pulmonary reconstruction with a single patch, and (3) when neonatal repair is not feasible, short-term pulmonary artery banding. When defects other than pulmonary stenosis are present, they occur generally soon after initial ASO, and reoperation is indicated whenever technically feasible and medical therapy fails to control the cardiovascular condition. Aortic valve insufficiency remains, however, a serious long-term complication that necessitates careful medical follow-up. A precise structural analysis of the neo-aortic root is essential to develop adequate surgical techniques for repair.

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